# Partial resistance to late blight (Phytophthora infestans) in hybrid progenies of four South American Solanum species crossed with diploid S. tuberosum

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# Partial resistance to late blight (*Phytophthora infestans*) in hybrid progenies of four South American *Solanum* species crossed with diploid *S. tuberosum*

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**Abstract** Resistant genotypes of the diploid tuber-bearing South American species *Solanum arnezii* × *hondelmannii*, *S. berthaultii*, *S. leptophyes* and *S. microdontum* were crossed with three diploid genotypes of *S. tuberosum* that varied in resistance and maturity type. The progenies were field tested for 2 years for resistance to a complex race of *Phytophthora infestans*. A wealth of genetic variation for resistance was found in most of the progenies. At least two susceptibility groups could be distinguished in some progenies of *S. microdontum*. This could be explained by the presence of several major resistance genes in the wild parent and, unexpectedly, in the susceptible parent SH 82-44-111. In most of the wild parents and in the susceptible parent SH 77-114-2988 there appeared to be minor resistance genes. General combining ability effects were predominant; small specific combining ability effects were detected in some crosses of *S. microdontum*. Gene action appeared dominant in some crosses.

**Key words** Inheritance · *S. arnezii* × *hondelmannii* *S. berthaultii* · *S. leptophyes* · *S. microdontum*

## Introduction

Late blight is a serious disease of potato (*Solanum tuberosum*) caused by the fungus *Phytophthora infestans* (Mont.) de Bary. Potato cannot be cultivated without chemical protection from this disease (Turkensteen 1993) because the level of (partial) resistance is too low at present. Much higher levels of resistance are found in related *Solanum* species such as *S. demissum*. In this species, 11 major resistance genes, called *R* genes, have been identified, and some have been transferred to the cultivated potato. However, races of the pathogen with virulence for all *R* genes have appeared and are now widespread (Turkensteen 1993), and therefore *R* gene-based resistances are no longer of value. It has been argued that the *demissum* genes have evolved in response to challenges with the pathogen, since both *S. demissum* and *P. infestans* are assumed to have evolved in Mexico (Nelson 1975). Therefore, resistant *Solanum* species originating from South America are particularly interesting, since in these species co-evolution with *P. infestans* has probably not taken place and the resistance may be of a less specific and hence more durable nature.

The South American species *S. berthaultii* and *S. microdontum* have a high resistance to *P. infestans*, whereas *S. arnezii* × *hondelmannii* and *S. leptophyes* have a moderate resistance (Colon and Budding 1988). For breeding purposes it is important to know how these resistances are inherited. At this moment, little is known about the inheritance of resistance in these species.

In *S. tuberosum* and *S. demissum* the inheritance of partial resistance to *P. infestans* has been studied. Partial resistance to *P. infestans* in *S. tuberosum* is usually considered to be a polygenic character (Ross 1986; Wastie 1991). Mapping of quantitative trait loci (QTL) suggests the involvement of at least seven chromosome regions (Leonards-Schippers et al. 1994). Some of the partial resistances in potato to *P. infestans* that were derived from *S. demissum* have been found to be based on single genes that have an incomplete effect. The resistance conferred by these partial *R* genes, *R2*, *R4*, *R10* and *R11* (Turkensteen 1993), may easily be mistaken, due to its partial nature, for oligo- or polygenically inherited partial resistance, unless tests are made with proper differentiating isolates of the pathogen or genetic analysis is done to estimate the number of genes involved. The occurrence of single genes conferring partial resistance indicates that partial resistance and polygenic inheritance, at least in the potato - late blight interaction, are by no means synonymous.

In this paper the inheritance of partial resistance to *P. infestans* is studied by means of field experiments for *S. arnezii* × *hondelmannii*, *S. berthaultii*, *S. leptophyes* and *S. microdontum* using hybrid progenies with diploid *S. tuberosum*.

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**Table 1** Accession names, gene bank (BGRC) codes and genotype designations of wild *Solanum* genotypes crossed with susceptible diploid *S. tuberosum*, average infection by *Phytophthora infestans* (area under the disease progress curve=ADPC; not transformed) and progeny sizes. ADPC values of Dutch *S. tuberosum* cultivars tested in the same experiments varied between 0.29 (most resistant) and 0.73 (most susceptible)

| Wild *Solanum* parent | | | | *S. tuberosum* parent | | |
|---|---|---|---|---|---|---|
| | | | | SH 82-44-111 (early) ADPC=0.57 | SH 77-114-2988 (late) ADPC=0.50 | SH 82-59-223 ADPC=0.32 |
| Species | BGRC code | Genotype | ADPC | Number of genotypes | Number of genotypes | Number of genotypes |
| *S. berthaultii* | 10.063 | *ber* 24 | 0.02 | 92 | 61 | 62 |
| *S. berthaultii* | 10.063 | *ber* 29 | 0.20 | 63 | 52 | 31 |
| *S. arnezii* × *hondelmannii* | 27.308 | *axh* 58 | 0.42 | –[a] | 42 | 53 |
| *S. leptophyes* | 17.196 | *lph* 81 | 0.36 | 43 | 37 | 35 |
| *S. microdontum* | 24.981 | *mcd* 167 | 0.01 | 75 | 49 | 68 |
| *S. microdontum* | 24.981 | *mcd* 178 | 0.09 | 85 | 77 | 72 |
| *S. microdontum* var. *gigantophyllum* | 18.570 | *mcd* 231 | 0.00 | 78 | 68 | 40 |
| *S. microdontum* var. *gigantophyllum* | 18.570 | *mcd* 264 | 0.07 | 67 | 61 | 14 |

[a] Cross not available

## Materials and methods

### Plant material

Wild parents for genetic analysis were chosen on the basis of a 3-year average rating of field infection by complex races of *P. infestans*, expressed as area under the disease progress curve (ADPC, see below). Of *S. berthaultii*, *S. microdontum* and *S. microdontum* var. *gigantophyllum*, two genotypes, one highly resistant and one partially resistant, were used. Of *S. arnezii* × *hondelmannii* and *S. leptophyes*, which are only moderately resistant, we used the most resistant genotype. Accession and genotype numbers, and average ADPC values of the parents are given in Table 1. All wild parents are diploids.

The wild parents were crossed with two susceptible diploid *S. tuberosum* CPRO-DLO-clones, SH 77-114-2988 (late) and SH 82-44-111 (early). To transfer the resistance to *S. tuberosum* breeding material, they were also crossed with SH 82-59-223, a diploid genotype combining several desirable characters such as 2n gametes, earliness and partial resistance to late blight; the latter progenies supplied additional information on inheritance. *S. tuberosum* was used as pollinator except in crosses with *S. berthaultii*, which only succeeded with *S. berthaultii* as male parent. Crosses were made during the summer in a greenhouse, seeds were extracted from the mature berries, dried and stored.

In the spring, seeds were pretreated with gibberellic acid (2 mg/l) for 24 h and sown in seed trays in sterilized soil. Seedlings were transplanted in pots, and grown to maturity to produce tubers. Tuber stocks were multiplied and annually renewed through cultivation in the greenhouse under natural short-day conditions. The tubers were stored at 4°C. If necessary, tuber dormancy was broken with Rindite (Burton 1989). The numbers of progeny genotypes tested for resistance are given in Table 1.

### Fungal material

*P. infestans* races 1.2.3.4.5.7.10.11 and 1.2.3.4.5.6.7.10.11 were provided by the DLO-Research Institute for Plant Protection (IPO-DLO), Wageningen, from liquid nitrogen-preserved stocks. The isolates were cultured on detached leaves of the susceptible cv 'Bildtstar' or moderately susceptible cv 'Nicola' at 15°C and 100% RH under continuous low-intensity fluorescent tube (Sylvania 'cool white' 40 W tubes) illumination.

Inoculum was prepared by rinsing leaflets with the sporulating fungus in tap water. The sporangial suspensions were placed at 10°C for 1–2 h to induce the release of zoospores. Spore densities were recorded by ten counts of 3.2 $mm^3$ samples of inoculum using a haemocytometer.

### Assessment of resistance in the field

The resistance of the wild parents was measured in the field over 3 years (1986–1988) as described in Colon and Budding (1988) using race 1.2.3.4.5.7.10.11 in 1986–1987 and race 1.2.3.4.5.6.7.10.11 in 1988. The resistance of the progenies to race 1.2.3.4.5.6.7.10.11 was measured in the field in the same way in 2 other years (1990 and 1991). Unfortunately, the parents could not be included with the progenies due to insufficient numbers of seed tubers. As standard cultivars, 'Bildtstar' (susceptible and medium-early to medium-late), 'Pimpernel' (partially resistant and late), 'Eersteling' (susceptible first early) and 'Apollonia' (1986–1988) or 'Ostara' (1990–1991; both moderately susceptible and early), all supposedly free of *R* genes, were used. These standard genotypes served to evaluate the evenness of the distribution of disease across the field and to compare the level of resistance of the progenies with those of known potato genotypes. During the second year in which the progenies were tested, the three diploid *S. tuberosum* parents were added to the set of standards.

Each year the trial, situated on sandy soil in the vicinity of Wageningen, consisted of two randomized blocks (Colon and Budding 1988, their Fig. 1). Four-plant plots of the wild parental genotypes were completely randomized within blocks among a larger set of wild *Solanum* materials. One two-plant plot of each progeny genotype was planted in each block and treated as a single experimental unit. Every ninth plot was used for one of the standard genotypes, which appeared in a fixed order. Seed tubers were planted at a distance of 0.35 m in hills that were 0.75 m apart. Planting was done in the last week of April. Patoran (metobromuron) was applied as a pre-emergence herbicide, and Imidan (fosmet) was applied against Colorado potato beetles. No other pesticides were applied.

Inoculations were made when the crop had fully developed, in the first half of July. Plants and soil were thoroughly soaked prior to inoculation, and the plants were inoculated late in the evening by spraying a spore suspension across the plots using a spraying arm with six nozzles, 0.75 m apart, that was connected to a propane tank at 2.5 bar. This was moved across the plots at a fixed speed of about 5 km/h. Border rows were not inoculated. In this way, about 50 $l\,ha^{-1}$

of inoculum was applied. Inoculum densities for the wild parents were $5 \times 10^4$ zoospores per milliliter. Inoculum densities for the progenies were $2.6 \times 10^4$ sporangia and $0.7 \times 10^4$ zoospores per milliliter in the first year and $1.7 \times 10^4$ sporangia and $1.0 \times 10^4$ zoospores per milliliter in the second year.

After inoculation, the trial field was irrigated in the mornings and evenings with a total of 8 l of water per square meter each day using a sprinkler system in order to improve the conditions for sporulation and infection by increasing the humidity.

Disease assessments were made at weekly intervals. The percentage of leaf area covered by late blight lesions was estimated for each plot using the most detailed of the two scales given by Colon and Budding (1988). From these readings the area under the disease progress curve (ADPC) was calculated according to the method of Shaner and Finney (1977) and the area was normalized as described by Fry (1978). In addition, the ADPC values were arcsin-sqrt-transformed to achieve normality.

### Statistical analyses

The trait under study, partial resistance to late blight, was expected to vary quantitatively, its variation resulting from segregating genes and also from environmental noise. For such a trait, the theory of classical quantitative genetics describes the genetics in terms of variances (Bulmer 1985). Therefore, variance components models were fitted to estimate variance components for year, blocks within years, genotype and the interaction between year and genotype using REML (Genstat 5 Committee 1987). Components larger than twice their standard error were considered significant.

In a segregating population, a quantitative trait may be considered to follow a mixture of (normal) distributions. Therefore, the likelihood of the normal model may be compared to the likelihood of normal mixture models with two or more underlying components to assess the segregation of major genes. To this end, mixture models with two underlying, presumably genetically based, components were fitted to the ADPC values of segregating populations. Population sizes were too small to allow for proper fitting of more extended mixture models. Calculations were carried out in Genstat (Genstat 5 Committee, 1987; Jansen 1994). Before fitting, ADPC values were adjusted for effects of year and block and also for interaction effects between genotype and year. For testing a normal model versus a normal mixture model with two components, a test statistic of twice the difference in the log-likelihoods was used (i.e. the likelihood ratio test statistic), which approximately follows a $\chi^2$-distribution with two degrees of freedom (Titterington et al. 1985). The results should still be regarded as preliminary; they have to be confirmed by further experiments.

To assess the general combining ability (GCA) and the specific combining ability (SCA) in the crosses, we fitted the model $E(y_{ij})=m+g_i+g_j+s_{ij}$ to the adjusted ADPC values, where m is the overall mean, $g_i$ is the main effect of parent i on the offspring, $g_j$ is the main effect of parent j on the offspring and $s_{ij}$ is the interaction effect between the parents (Bulmer 1985). Crosses differed in variability, and therefore a weighted analysis of variance was done using as a weight the reciprocal of the residual variance within each cross.

Progeny means were predicted from the model used to estimate GCA and SCA. For each progeny, linear correlations were estimated between the genotypic means of transformed ADPC of the 2 years.

Calculations were done with Genstat (Genstat 5 Committee 1987). Significance levels were $P=0.05$, unless stated otherwise.

## Results

Most progenies exhibited a wide genetic variation, since genotypic effects were significant, while year effects and genotype × year and year × block interactions were not (Tables 2 and 3). Correlations between years for progenies with a significant genotype effect ranged from $r=0.29$ to $r=0.86$, and averaged $r=0.60$. The genotypic component was especially large in the progenies of *mcd* 167 and *mcd* 178. The distribution patterns of resistance levels differed widely (Figs. 1–3). Some of the progenies with a significant genetic component, especially those of *mcd* 167 and *mcd* 178, segregated into a wide range of phenotypes, while others, like those of *ber* 29 and *lph* 81, had a narrow distribution. The progenies of SH 77-114-2988 were generally segregating into a wider range, and their mean ADPCs

**Table 2** Estimates of components of variance (vcomp) for ADPC in progenies of *ber* 24, *ber* 29, *axh* 58 and *lph* 81 crossed with SH 82-44-111, SH 77-114-2988 and SH 82-59-223, assessed in the field against race 1.2.3.4.5.6.7.10.11 of *P. infestans*. Components larger than twice their standard error may be considered to be significant

| | | SH 82-44-111 | SH 77-114-2988 | SH 82-59-223 |
|---|---|---|---|---|
| | | vcomp ± SE | vcomp ± SE | vcomp ± SE |
| *ber* 24 | Year | 0.2 ± 4.6 | 1.8 ± 6.3 | 0.0 |
| | Year.block | 4.7 ± 5.0 | 4.0 ± 4.5 | 4.1 ± 3.8 |
| | Genotype | 2.5 ± 2.3 | 12.1 ± 4.7 | 6.3 ± 2.7 |
| | Genotype.year | 2.7 ± 3.2 | 3.8 ± 4.3 | 0.0 |
| | Residual | 27.4 ± 3.1 | 26.2 ± 3.9 | 19.3 ± 2.7 |
| *ber* 29 | Year | 2.2 ± 4.5 | 18.8 ± 31.1 | 0.0 |
| | Year.block | 1.0 ± 1.8 | 4.2 ± 5.3 | 0.0 |
| | Genotype | 11.0 ± 4.6 | 30.8 ± 11.9 | 0.0 |
| | Genotype.year | 0.0 | 10.7 ± 9.1 | 16.8 ± 7.2 |
| | Residual | 36.4 ± 4.7 | 35.6 ± 6.3 | 22.5 ± 5.5 |
| *axh* 58 | Year | | 12.2 ± 28.4 | 0.0 |
| | Year.block | | 12.6 ± 13.6 | 4.0 ± 4.2 |
| | Genotype | | 29.5 ± 11.8 | 32.4 ± 10.9 |
| | Genotype.year | | 9.4 ± 8.5 | 8.4 ± 7.4 |
| | Residual | | 29.3 ± 5.6 | 30.5 ± 5.3 |
| *lph* 81 | Year | 0.0 | 5.0 ± 15.2 | 5.2 ± 10.6 |
| | Year.block | 12.0 ± 11.4 | 8.6 ± 9.4 | 1.5 ± 4.2 |
| | Genotype | 0.2 ± 10.1 | 18.3 ± 8.9 | 0.0 |
| | Genotype.year | 23.8 ± 13.6 | 9.5 ± 7.1 | 0.0 |
| | Residual | 39.6 ± 7.7 | 20.0 ± 4.2 | 58.1 ± 9.0 |

**Table 3** Estimates of components of variance (vcomp) for ADPC in progenies of *mcd* 167, *mcd* 178, *mcd* 231 and *mcd* 264 crossed with SH 82-44-111, SH 77-114-2988 and SH 82-59-223, assessed in the field against race 1.2.3.4.5.6.7.10.11 of *P. infestans*. Components larger than twice their standard error may be considered to be significant

| | | SH 82-44-111 | SH 77-114-2988 | SH 82-59-223 |
|---|---|---|---|---|
| | | vcomp ± SE | vcomp ± SE | vcomp ± SE |
| *mcd* 167 | Year | 1.1 ± 2.4 | 0.0 | 3.8 ± 6.7 |
| | Year.block | 0.7 ± 1.2 | 0.8 ± 1.5 | 1.0 ± 1.4 |
| | Genotype | 109.4 ± 19.5 | 157.4 ± 35.5 | 64.9 ± 13.5 |
| | Genotype.year | 1.2 ± 3.3 | 7.8 ± 6.8 | 6.6 ± 4.1 |
| | Residual | 29.5 ± 3.6 | 39.9 ± 6.0 | 22.0 ± 3.0 |
| *mcd* 178 | Year | 1.4 ± 3.9 | 0.0 | 0.0 |
| | Year.block | 2.0 ± 2.3 | 0.5 ± 1.0 | 2.7 ± 2.9 |
| | Genotype | 60.2 ± 11.2 | 38.6 ± 9.3 | 28.7 ± 7.7 |
| | Genotype.year | 6.8 ± 3.6 | 10.0 ± 5.3 | 0.0 |
| | Residual | 25.1 ± 2.9 | 33.8 ± 4.1 | 54.0 ± 5.7 |
| *mcd* 231 | Year | 0.9 ± 1.6 | 0.0 | 0.0 |
| | Year.block | 0.0 | 1.5 ± 1.7 | 0.0 |
| | Genotype | 23.4 ± 4.7 | 26.7 ± 6.4 | 21.9 ± 7.0 |
| | Genotype.year | 0.0 | 0.0 | 0.0 |
| | Residual | 18.9 ± 1.9 | 35.1 ± 3.7 | 28.8 ± 4.1 |
| *mcd* 264 | Year | 0.8 ± 1.8 | 2.4 ± 4.3 | 0.0 |
| | Year.block | 0.2 ± 0.7 | 0.7 ± 1.3 | 0.0 |
| | Genotype | 7.8 ± 4.6 | 9.0 ± 3.2 | 12.5 ± 13.4 |
| | Genotype.year | 8.9 ± 5.1 | 0.0 | 0.0 |
| | Residual | 28.7 ± 3.9 | 25.3 ± 3.0 | 41.2 ± 13.6 |

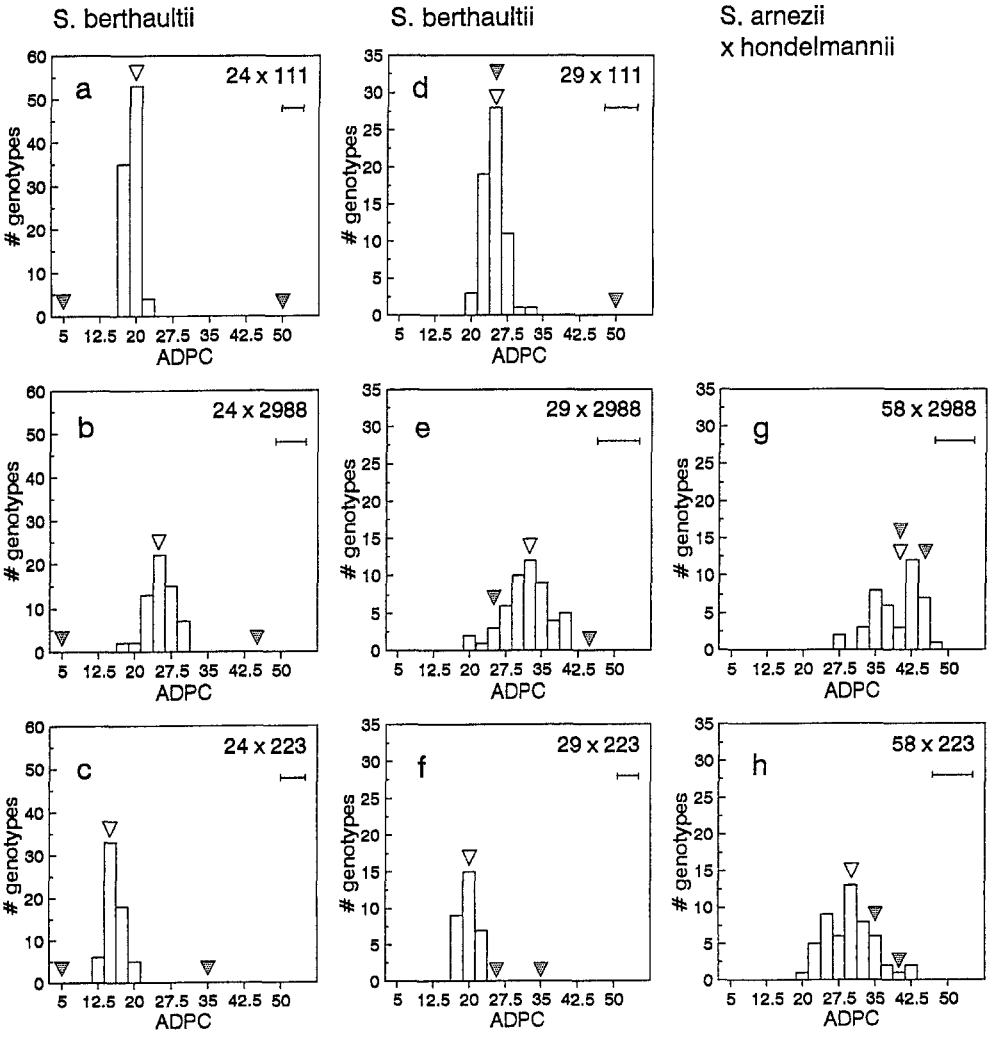


**Fig. 1a–h** Frequency distribution of average levels of resistance to *P. infestans* race 1.2.3.4.5.6.7.10.11 of progenies of *ber* 24 (**a–c**), *ber* 29 (**d–f**) and *axh* 58 (**g, h**) crossed with the susceptible diploid *S. tuberosum* genotypes SH 82-44-111 (**a, d**), SH 77-114-2988 (**b, e, g**) or SH 82-59-223 (**c, f, h**), assessed in the field over 2 years. Twenty-one groups are distinguished, on the basis of the average area under the disease progress curve (ADPC). The upper limit of each third ADPC interval is given at the x-axis. *Bars* indicate LSD at $P < 0.05$. Parents, tested with races 1.2.3.4.5.7.10.11 and 1.2.3.4.5.6.7.10.11 in 3 other years, are marked with *shaded arrowheads* and progeny means with *open arrowheads*

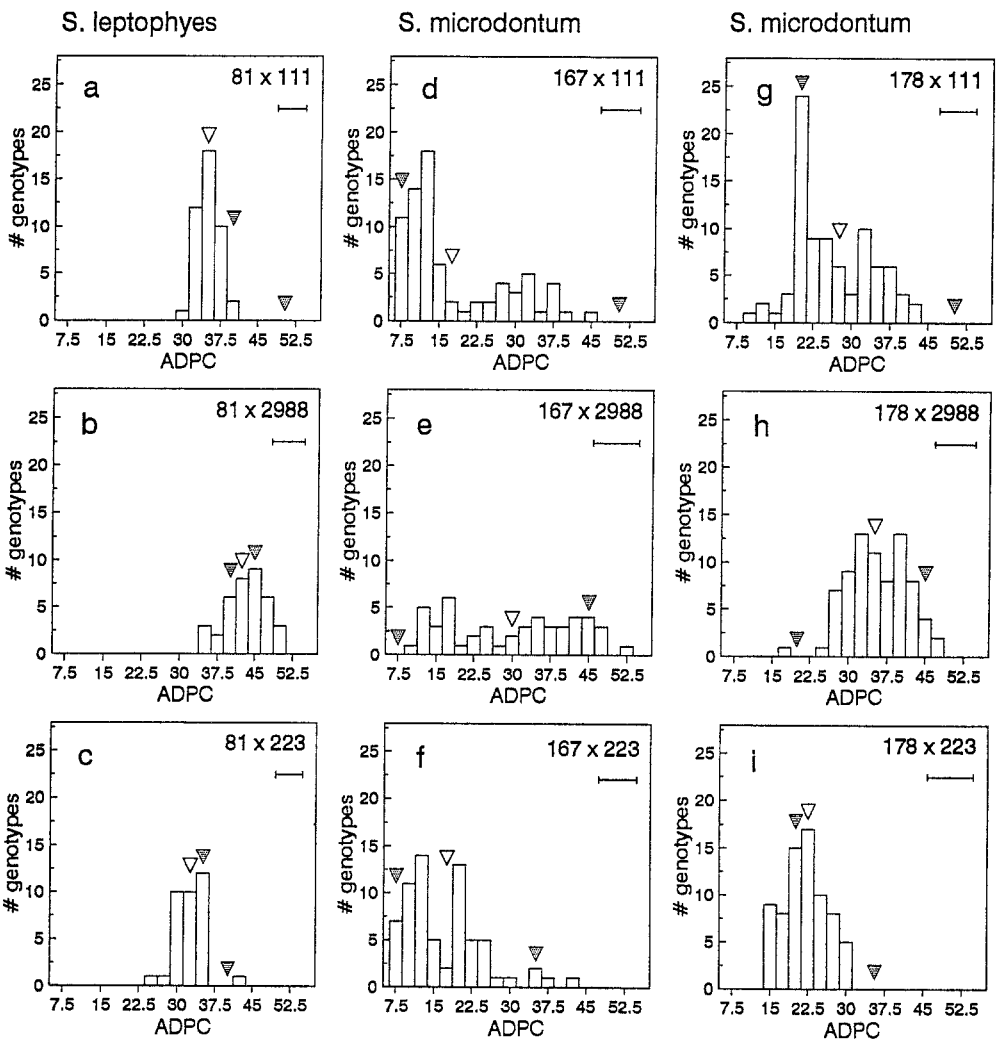


**Fig. 2a–i** Frequency distribution of average levels of resistance to *P. infestans* race 1.2.3.4.5.6.7.10.11 of progenies of *lph* 81 (**a–c**), *mcd* 167 (**d–f**) and *mcd* 178 (**g–i**) crossed with the susceptible diploid *S. tuberosum* genotypes SH 82-44-111 (**a, d, g**), SH 77-114-2988 (**b, e, h**) or SH 82-59-223 (**c, f, i**), assessed in the field over 2 years. Twenty groups are distinguished, on the basis of the average area under the disease progress curve (ADPC). The upper limit of each third ADPC interval is given at the x-axis. *Bars* indicate LSD at $P< 0.05$. Parents, tested with races 1.2.3.4.5.7.10.11 and 1.2.3.4.5.6.7.10.11 in 3 other years, are marked with *shaded arrowheads* and progeny means with *open arrowheads*

**Table 4** Segregation into one or two susceptibility groups in progenies of *mcd* 167, *mcd* 178 and *mcd* 231 crossed with *S. tuberosum* SH 82-44-111 (111), SH 77-114-2988 (2988) and SH 82-59-223 (223). Susceptibility was assessed as ADPC in the field against race 1.2.3.4.5.6.7.10.11 of *P. infestans*. The test statistic (see text) for the difference between the one-component model and the two-components model, and estimates for p, SE and $s^2$ for the most likely models are given. The percentage given with $s^2$ indicates how much it is reduced compared with that of the one-component model given in Tables 2 and 3 ($p_i$ proportion of genotypes in group i, $SE_i$ standard error of $p_i$)

| Cross | Test statistic | $p_1$ | $p_2$ | $SE_1$ | $SE_2$ | $s^2$ | (%) |
|---|---|---|---|---|---|---|---|
| | $1 \rightarrow 2$ | | | | | | |
| *mcd* 167 × 111 | 56.69 | 0.69 | 0.31 | 0.05 | 0.53 | 14.3 | (87) |
| *mcd* 167 × 2988 | 15.72 | 0.45 | 0.55 | 0.10 | 0.10 | 31.3 | (80) |
| *mcd* 167 × 223 | 12.62 | 0.55 | 0.44 | 0.10 | 0.20 | 31.0 | (52) |
| *mcd* 178 × 111 | 18.14 | 0.64 | 0.36 | 0.06 | 0.06 | 15.0 | (75) |
| *mcd* 178 × 2988 | 0.54 | 1.0 | – | – | – | 34.5 | (11) |
| *mcd* 178 × 223 | 1.73 | 1.0 | – | – | – | 18.0 | (37) |
| *mcd* 231 × 111 | 13.76 | 0.67 | 0.33 | 0.06 | 0.31 | 7.3 | (69) |
| *mcd* 231 × 2988 | 2.09 | 1.0 | – | – | – | 19.2 | (28) |
| *mcd* 231 × 223 | 18.69 | 0.97 | 0.03 | 0.00 | 0.00 | 7.6 | (65) |
| Significant at $P< 0.05$ if > | 6.0 | | | | | | |
| Significant at $P< 0.01$ if > | 9.2 | | | | | | |

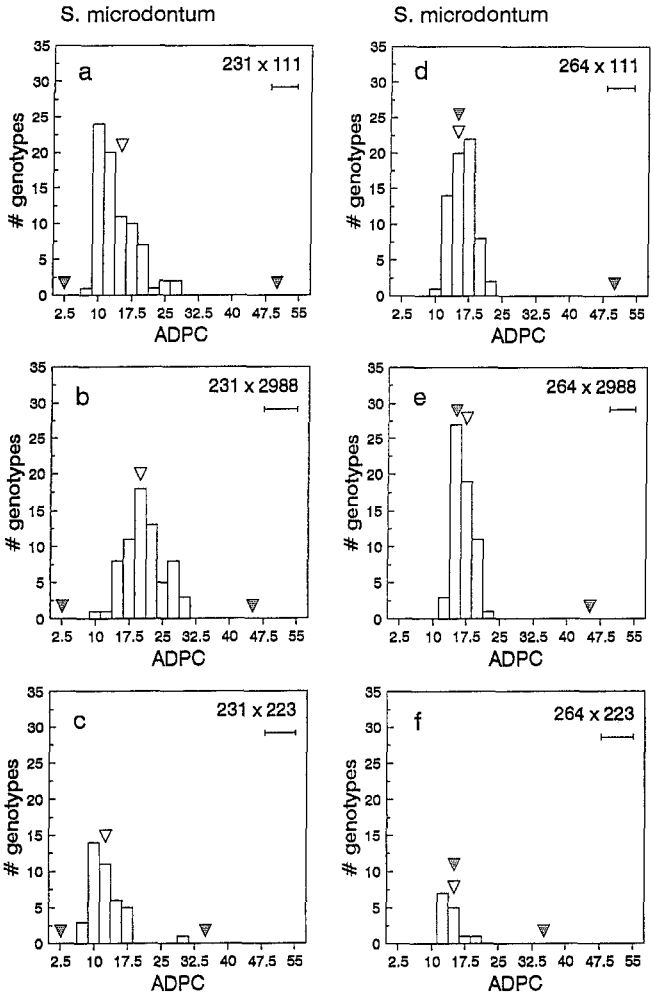


**Fig. 3a–f** Frequency distribution of average levels of resistance to *P. infestans* race 1.2.3.4.5.6.7.10.11 of progenies of *mcd* 231 (**a–c**) and *mcd* 264 (**d–f**) crossed with the susceptible diploid *S. tuberosum* genotypes SH 82-44-111 (**a, d**), SH 77-114-2988 (**b, e**) or SH 82-59-223 (**c, f**), assessed in the field over 2 years. Twenty-two groups are distinguished, on the basis of the average area under the disease progress curve (ADPC). The upper limit of each third ADPC interval is given at the x-axis. *Bars* indicate LSD at $P< 0.05$. Parents, tested with races 1.2.3.4.5.7.10.11 and 1.2.3.4.5.6.7.10.11 in 3 other years, are marked with *shaded arrowheads* and progeny means with *open arrowheads*

were higher (more susceptible) than those of SH 82-44-111 and SH 82-59-223. Transgression towards resistance, indicated by a significant difference between the mean ADPC of the most resistant progeny genotype and that of the most resistant parent, appeared to occur in the progenies of *axh* 58, in *mcd* 178 × SH 82-44-111 and in *mcd* 178 × SH 82-59-223. In some progenies, especially those of *mcd* 167 and *mcd* 178, the distributions appeared to contain more than one underlying component. No significant genotypic component was found in *ber* 24 × SH 82-44-111, in the progenies of *lph* 81 and *mcd* 264 with SH 82-44-111 and SH 82-59-223, and in *ber* 29 × SH 82-59-223. These progenies probably do not contain much genetic variation.

**Table 5** Mean ADPC of the suceptible cvs 'Bildtstar' and 'Eersteling', and the partially resistant cv 'Pimpernel' in the 5 years of the experiments

| Cultivar | Bildtstar | Eersteling | Pimpernel | Mean |
|---|---|---|---|---|
| Year | Mean ± SE | Mean ± SE | Mean ± SE | |
| 1986 | 57.5 ± 0.4 | 59.9 ± 0.5 | 42.1 ± 0.5 | 53.2 |
| 1987 | 51.1 ± 0.5 | 54.3 ± 0.5 | 38.2 ± 0.5 | 47.9 |
| 1988 | 55.6 ± 0.6 | 58.8 ± 0.5 | 48.5 ± 0.6 | 54.3 |
| 1990 | 53.2 ± 0.4 | 59.3 ± 0.4 | 33.0 ± 0.4 | 48.5 |
| 1991 | 51.2 ± 0.4 | 56.9 ± 0.5 | 32.4 ± 0.5 | 46.8 |
| Mean | 53.7 | 57.8 | 38.8 | |

**Table 6** Phenotypic means of area under the disease progress curve (ADPC) of the parents and progenies of *ber* 24, *ber* 29, *axh* 58, *lph* 81, *mcd* 167, *mcd* 178, *mcd* 231 and *mcd* 264 with SH 82-44-111, SH 77-114-2988 and SH 82-59-223, assessed in the field against race 1.2.3.4.5.6.7.10.11 of *P. infestans*. All means of crosses were predicted from the regression model used to estimate GCA and SCA effects. The wild parents were tested for resistance in 1986-1989, the progenies and the SH parents in 1990 and 1991

| Parents | Parental mean | SH 82-44-111 | SH 77-114-2988 | SH 82-59-223 | Mean of crosses |
|---|---|---|---|---|---|
| | | 49.1 | 45.0 | 34.3 | |
| *ber* 24 | 4.8 | 18.0 | 24.0 | 14.7 | 18.9 |
| *ber* 29 | 23.3 | 23.4 | 30.6 | 18.6 | 24.2 |
| *axh* 58 | 39.7 | – | 38.0 | 28.5 | – |
| *lph* 81 | 37.9 | 33.8 | 42.0 | 31.4 | 35.7 |
| *mcd* 167 | 5.6 | 16.4 | 28.5 | 15.6 | 20.2 |
| *mcd* 178 | 17.7 | 25.1 | 34.5 | 20.7 | 26.8 |
| *mcd* 231 | 1.2 | 12.9 | 19.7 | 11.3 | 14.6 |
| *mcd* 264 | 13.7 | 14.6 | 15.6 | 13.1 | 14.4 |
| Mean of crosses | | 21.2 | 29.1 | 19.2 | |

Significant segregations into two groups were detected in the progenies of *mcd* 167, *mcd* 178 and *mcd* 231 (Table 4). Progenies of these genotypes crossed with SH 82-44-111 segregated in a 3:1 (R:S) ratio, while those with SH 77-114-2988 and SH 82-59-223 either did not segregate into groups, or segregated in a 1:1 ratio. The susceptible component sometimes consisted of a wide range of phenotypes, and could significantly be split up further. However, these secondary separations were not very clear, as standard errors obtained for the estimated proportions were high (not shown). None of the other progenies significantly segregated into groups.

The GCA/SCA analysis resulted in mean sums of squares of $p_1$ (278.6, $df$=6) and $p_2$ (287.0, $df$=2) which were relatively large when compared with the interaction mean sum of squares (12.3, $df$=13), although all effects were significant at $P< 0.001$. Therefore, general combining ability appeared to be predominant, and the average performance of the crosses could reasonably be predicted from their parental values. Of the interaction mean sum of squares 60% was attributed to the cross *mcd* 264 × SH 77-114-2988.

Partially resistant cv 'Pimpernel' was significantly ($P< 0.001$) less affected by late blight than susceptible cvs

'Bildtstar' and 'Eersteling' in all years (Table 5). However, there was a year effect and a cultivar × year interaction that were both significant at $P< 0.001$, though small compared to the effect of the cultivars.

The phenotypic means of most progenies of SH 82-44-111 and SH 82-59-223 were lower than those of the corresponding progenies of SH 77-114-2988 (Table 6). The exception to this were the crosses of *mcd* 264, all of which had about the same genotypic mean, which appeared to equal the mean of the parent *mcd* 264. In the progenies of *ber* 29, *axh* 58 and *lph* 81 with SH 82-59-223 and in *lph* 81 × SH 82-44-111, progeny means were lower than both parental means, indicating transgression towards resistance.

## Discussion

A wealth of genetic variation for resistance to *P. infestans* was found in most of the progenies of the four South American *Solanum* species crossed with diploid *S. tuberosum*. Some of the progenies significantly segregated into resistant and susceptible groups. Therefore, part of the genetic variation might be contributed to major genes, conferring partial rather than complete resistance, in several of the wild parents under investigation. The action of minor genes appears also to be present, as genetic variation was found in most progenies that did not significantly segregate into groups.

Most of the genetic variation was found in the progenies of *mcd* 167, *mcd* 178, *axh* 58 and *mcd* 231. The progenies of the other wild parents varied much less. With respect to the *S. tuberosum* parents, SH 77-114-2988 gave more variable progenies than the other two SH parents.

A comparison of the parental and progeny means of the crosses to find out whether dominant genes are present in the crosses was not possible because parents and progenies were not tested in the same years; our findings that progeny means are closer to that of the wild parent than to that of the SH parent may also be due to differences between years. A general influence of years on resistance for all these wild species and slight genotype × year interactions for *S. microdontum*, have already been demonstrated (Colon and Budding 1988). The isolate used to test the resistance of the progenies differed from the one used in the first 2 years of parental testing. The standard cultivar 'Pimpernel' in particular had a higher ADPC in the years when the wild parents were tested than when the SH parents and the progenies were tested. It has to be taken into account that only when parents and progenies are tested simultaneously and with the same isolate can additive effects be accurately distinguished from dominant effects.

General combining ability appeared important; the parents in general, rather than specific combinations determine the resistance of the progeny. However, no conclusions can be drawn as to how genes from the different wild *Solanum* species would combine in interspecific crosses between the wild *Solanum* species.

### *S. microdontum*

By far the most genetic variation occurred in the progenies of *mcd* 167, *mcd* 178 and *mcd* 231, while those of *mcd* 264 were rather uniform. We have evidence that major genes appeared to segregate in some of these progenies.

The segregations in the crosses of *mcd* 167, *mcd* 178 and *mcd* 231 with SH 82-44-111 can be explained by the segregation of two genes per cross, one in each of the wild parents and the other in SH 82-44-111. The gene in *mcd* 178 and that in *mcd* 231 in this model have to have a relatively small effect, since the separation in groups was not significant in crosses with SH 77-114-2988 and SH 82-59-223. However, this model does not explain the single, susceptible genotype segregating in mcd 231 × SH 82-59-223. This genotype may be a rare segregant that happens to be present in this progeny but missing in the other progenies.

In the crosses with *mcd* 264, no major genes appeared to segregate. The parent *mcd* 264 may have at least one homozygous major gene epistatic over the genes from the SH parents, as *mcd* 264 × SH 77-114-2988 was as resistant as *mcd* 264 × SH 82-44-111 and *mcd* 264 × SH 82-59-223, while with the other wild parents the crosses with SH 77-114-2988 were more susceptible than those with the other two SH parents. The relatively high resistance of *mcd* 264 × SH 77-114-2988 accounts for most of the interaction term in the GCA/SCA analysis.

Although parental and progeny means cannot be compared directly, some of the phenotypic means, like those of the non-segregating progeny of *mcd* 264 or of the resistant groups in the progenies of *mcd* 167 and *mcd* 231, are so close to that of the resistant parent that dominant gene action may be assumed. Backcross populations should be made and compared with their parents to see whether these genes are really dominant.

### *S. berthaultii*

With respect to *S. berthaultii*, the results suggest that the two wild parents differ in resistance genes since the progenies of *ber* 24 were more resistant than the corresponding progenies of *ber* 29. The parents *ber* 24 and *ber* 29 may have homozygous major genes, since their progenies did not seem to segregate.

The widest genetic variation was found in the crosses with SH 77-114-2988. The finding that the variation in crosses with the other SH parents is much smaller suggests that most of the genetic variation in the crosses with SH 77-114-2988 came from the SH parent. In crosses with SH 82-44-111 and SH 82-59-223, the progeny means were lower (more resistant) than with SH 77-114-2988. This suggests gene action from these two SH parents rather than from *S. berthaultii*.

### *S. arnezii* × *hondelmannii*

The two progenies of *axh* 58 had a wider range of phenotypes than those of *S. berthaultii*. Therefore, minor genes

for resistance appear to be present in *axh* 58. These genes add to the effect of genes from *S. tuberosum*, since transgression towards resistance was found. The higher resistance of *axh* 58 × SH 82-59-223 compared with *axh* 58 × SH 77-114-2988 suggests that SH 82-59-223 donated genes for resistance in the first cross.

*S. leptophyes*

The progenies of *lph* 81 resembled those of *ber* 24 and *ber* 29 with respect to the genetic variation derived from the wild parents and from the SH parents, except that the resistance level was generally much lower. The progenies with SH 82-44-111 and SH 82-59-223 were slightly more resistant than those with SH 77-114-2988, and appeared to show transgression towards resistance. This again suggest a contribution by resistance genes from SH 82-44-111 and SH 82-59-223. The parent *lph* 81 does not appear to have donated significant resistance to its progenies.

The results discussed above strongly suggest that not only the wild species but also one of the susceptible *S. tuberosum* parents is likely to carry major genes for resistance. The segregation ratios of 3:1 (R:S) instead of 1:1 in the progenies of *mcd* 167, *mcd* 178 and *mcd* 231 crossed with SH 82-44-111 suggests that two genes conferring partial resistance are involved. The different ratios in the progenies of SH 77-114-2988 and SH 82-59-223 crossed with these wild parents suggest that at least one segregating gene must be from SH 82-44-111. The putative single gene from SH 82-44-111 appears to have a major effect on the resistance of some progenies and a minor effect in others. However, it does not confer resistance to SH 82-44-111. This suggests an epistatic gene action of this gene with genes from the wild parents. Evidence for epistatic gene action has been found for *R* genes from *S. demissum* in crosses between diploid *S. tuberosum* (A. El-Kharbotly, Wageningen Agricultural University, personal communication). It is not known whether *S. demissum* is involved in the ancestry of the *S. tuberosum* parents that we used. The relatively wide variation, which appears to be of genetic origin, in crosses with SH 77-114-2988 suggests the segregation of only minor genes from this SH parent. The number of (heterozygous) major and minor genes cannot be deduced from the data presented here but may be derived from linkage analysis with restriction fragment length polymorphisms or other molecular markers to map quantitative trait loci (Lander and Botstein 1989) for resistance on the genomes of these *Solanum* species, for which the populations discussed may be used.

We have demonstrated that high levels of partial resistance are transferred to the progeny when partially resistant *Solanum* genotypes are crossed with *S. tuberosum*. Transferring this resistance to the cultivated potato should be possible and could be done by repeated backcrossing with *S. tuberosum*, under selection for resistance, with regular intercrossing to assemble all resistance genes in one genotype before the next backcross is done. More than one backcross is probably necessary to improve characters such as daylength sensitivity, yield, stolon length and others.

Although the resistance coming from Bolivian and Peruvian species, rather than Mexican Solanaceae, is hoped to be durable, the great effort required to introduce this resistance into cultivars makes it necessary to assess the durability of this resistance, for example by means of comparing the resistance mechanism with that of resistance conferred by *R* genes.

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